March 2, 2012

Connecticut General Assembly Environment Committee
Room 3200, Legislative Office Building
Hartford, CT 06106

Dear Senator Meyer, Representative Roy, Senator Roraback, Representative Chapin and Members of the Environment Committee,

I am writing, as a representative of Central Life Sciences, to express our opposition to HB 5260 —which would restrict the use of certain products used for mosquito control in waterways, catch basins and storm drains in Connecticut.

Of particular concern is the restriction of methoprene, the active ingredient in Altosid®, a product manufactured by Central Life Sciences and trusted since it was first registered with the U.S. Environmental Protection Agency (EPA) in 1975 as a valuable tool in mosquito abatement programs. These programs are vital to maintaining and protecting the public from the spread of serious mosquito-borne disease like West Nile virus and Eastern Equine Encephalitis.

Many laboratory and field studies have been performed to determine any impacts of methoprene on the environment. Numerous studies have been conducted that consistently demonstrate that methoprene is either undetectable or present at extremely low concentrations when used at recommended application levels. Since it was first registered with the EPA in 1975, and reviewed again in 1992, methoprene has been repeatedly tested for safety and effectiveness under EPA guidelines, which includes testing on a wide range of crustaceans.

Methoprene *does not* bioaccumulate in fish. It *does not* persist in the environment. But it does perform a crucial role in killing mosquito larva, thereby protecting the public from the diseases carried by adult mosquitoes.

Methoprene is even exempted from the requirement of food tolerances by the EPA. Thus, it may be applied to all raw agricultural commodities for human consumption and is approved by the World Health Organization for application to drinking water where mosquitoes may breed and spread disease.

While we recognize the challenges and frustrations that lobstermen in the Long Island Sound are facing and understand the emotion attached to the issue, we urge you to base any decisions regarding methoprene on sound science and the available research.

It is our sincere hope that you will commit to examining this issue further before prematurely banning a product that has been trusted for years to help protect and uphold public health, including the citizens of Connecticut. We thank you for giving us the opportunity to present this information. We look forward to continuing to work with you in the future, and our company stands ready to provide any additional information you might need.

Sincerely,

Mark Newberg
Director, Corporate Affairs and Communications
Central Life Sciences

METHOPRENE: A TESTED, TRUSTED TOOL IN PROTECTING PUBLIC HEALTH

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SUMMARY: METHOPRENE AND MOSQUITO CONTROL

More than 40 years ago, Central Life Sciences discovered insect growth regulators (IGR) which paved the way for biorational products, i.e. products derived from a variety of biological sources. These products prevent the development of nuisance and disease-carrying insects—like mosquitoes—before they can cause economic damage and spread dangerous illnesses like West Nile virus and Eastern Equine Encephalitis (EEE). IGRs are the active ingredients in many of the products Central Life Sciences manufactures today.

Altosid®, a larvicide from Central Life Sciences, is commonly used by mosquito abatement districts and public health department officials, and contains methoprene. The methoprene in Altosid stops mosquitoes from becoming flying, breeding, biting adults. Methoprene is target-specific and will not affect fish, waterfowl, mammals or beneficial predatory insects. As a result, vector control professionals can feel comfortable and confident using Altosid in their district's most sensitive areas — and feel even better about the effective, long-term control provided in return. Furthermore, due to the effectiveness and residual larvicidal activity, the use of Altosid reduces adult mosquito populations and the need for wide-spread spraying of products into the environment.

First registered by the Environmental Protection Agency (EPA) in 1975 as a biorational product, in later years, after the EPA reorganized divisional branches, Altosid fell under the jurisdiction of the Biopesticide Division.

Several formulations of Altosid (liquid, pellet, or briquette) have been developed for specific mosquito larvicidal applications depending on the particular usage. All of the formulations are designed to maintain a low, yet effective concentration of the active ingredient at the desired treatment site through the sensitive late fourth larval instar, or developmental stage, of the mosquito. Applications of Altosid to larval mosquito habitat through this stage prevent the development of and subsequent emergence of adult mosquitoes.

The EPA classifies methoprene in toxicity categories III and IV, which means slightly toxic to practically nontoxic. The EPA's Methoprene Fact Sheet states that study data indicate "oral, dermal or inhalation exposure to methoprene for an extended duration is not likely to cause adverse health effects in humans."

Extensive research has clarified methoprene's effects on non-target aquatic organisms, including crustacea. Studies of "immature and adult anthropods demonstrate 24- and 48- hour LC_{50} values [that is, the median lethal concentration — the concentration predicted to kill 50 percent of the organism] — is greater than 900 ppb [parts per billion]." Additional studies demonstrate no sensitivity of larvae to methoprene at concentrations up to 1,000 ppb. These concentrations are approximately 500 times higher than the concentration of methoprene (0.2 ppb) needed to control mosquitoes.

The EPA concluded in its review that "all the environmental fate data requirements for methoprene have been satisfied." Furthermore, methoprene degrades rapidly in water, is metabolized rapidly in soil, and does not leach; therefore it "is not expected to persist in soil or contaminate ground water."

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SECTION I: METHOPRENE IS TRUSTED AND HAS BEEN TESTED

Many laboratory and field studies have been performed to study impact of methoprene on the environment. Studies consistently demonstrate that methoprene is either undetectable or present at extremely low concentrations when used at recommended application levels.

Since it was first registered with the EPA in 1975, and reviewed again in 1992, methoprene has been repeatedly tested for safety and effectiveness under EPA guidelines, which includes testing on a number of crustaceans.

METHOPRENE IS TARGET SPECIFIC TO MOSQUITOES AND A FEW OTHER INSECTS

The favorable environmental properties of methoprene have been established by the work of many different research groups. If used as recommended, there is little to no effect on non-target species like mammals, fish, crustaceans, birds, protozoa, annelids, mollusks, amphibian, damsel flies, mayflies, water beetles or waterfowl.

The toxicity of methoprene to mammals has been studied extensively. The mammalian LD_{50}, or median lethal dose, for methoprene is greater than 34,560 mg/kg body weight. Methoprene does not cause gene mutations or cancer. It does not cause birth defects and thus, is considered a less toxic approach for insect control. Methoprene and other IGRs are used to control pests in stored grain, fleas on companion animals, flies in cattle production and other areas where insects can spread disease and cause economic damage.

Methoprene is exempted from the requirement of food tolerances by EPA; therefore it may be applied to all raw agricultural commodities for human consumption. It is also approved by the World Health Organization for application to drinking water where mosquitoes may breed and spread disease.

METHOPRENE DOES ITS JOB THEN GOES AWAY

Methoprene does not persist in the environment after use and is subject to degradation from UV light and metabolism in plants, animals, aquatic microorganisms and soil microbes[1]. It is stable in water for only a few hours and in cases of heavy microbe populations or full sun exposure, the persistence is further reduced.

This lack of persistence is one reason that slow release formulations were developed to provide the methoprene concentration necessary to control mosquitoes. As methoprene degrades, the slow release formulations provide a continuous source of active ingredient at the desired effective rate into the water column until the formulation is exhausted. Even in these slow-release formulations, methoprene does not bioaccumulate in fish and does not persist in the environment.

[1] Schaeffer and Dupras Jr. 1973, Quistad et al. 1974a, 1974b, 1975a, 1975b, 1975c, 1975d, 1976a, 1976b; Chamberlain et al. 1975, Schooley et al. 1975a, 1975b; Schooley and Quistad 1979.

SECTION II: CLAIMS AGAINST METHOPRENE HAVE BEEN WIDELY DISPROVED

CLAIM: Methoprene is highly toxic to lobsters at low concentrations.
TRUTH: Methoprene is not toxic to lobsters at concentrations found in waters surrounding treated areas. Conflicting data have been reported on the toxicity of methoprene to lobsters.

- In the *Journal of ShellfishResearch*, Zulkosky et al reported that methoprene at 10 ppb caused no toxicity in Stage I-II larval lobsters.[2]

- Hans Laufer, as detailed in a speech at the 2003 Long Island Sound Lobster Health Symposium, said that 1 ppb, 10 ppb, and 100 ppb concentrations of methoprene did not delay stage transitions in Stages I-III, though higher concentrations did.[3]

- Walker et al reported in the *Journal of Shellfish Research* "that Stage IV larvae exposed to methoprene concentrations of 50 ppb experienced a greater than 90 percent mortality rate after three days." [4] A similar study found that methoprene was toxic to State II larvae at 1 ppb and Stage IV larvae at 5 ppb. In context, the recommend rate of application for methoprene in vector control programs is 0.2 ppb.

CLAIM: Maine does not use methoprene and they have abundant lobster populations.
TRUTH: According to Maine's Supervisor of Mosquito and Tick Control, both larvicides methoprene and BTI are used to control mosquitoes in standing water in Maine. Additionally, there is no scientific basis for any connection between Maine's use of methoprene and their abundant lobster population, which can be linked to various environmental factors, including water temperature.

- In discussions with the Maine's Supervisor of Mosquito and Tick Control, both larvicides methoprene and Bti are used to control mosquitoes in standing water. Their use is restricted to contained waters that do not move into streams or creeks to avoid movement of chemicals from one private property to another.

- Environmental conditions in Maine make waters much more hospitable to lobsters. Water temperature is much colder north of Nantucket Sound. Robert Glenn, a senior marine fisheries biologist with the state Division of Marine Fisheries said in a 2010 article in the *Cape Code Times,* that "Although temperature increases have been noted in Boston and in Maine, the water is so much colder north of Nantucket Sound that it would take a huge increase in the average temperature, of 15 degrees or more, to reach levels that lobsters can't tolerate."[5]

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[2] Zulkosky, A. et al. "Acute toxicity of resmiethrin, malathion and methoprene to larval and juvenile American lobsters (Homarus americanus) and analysis of pesticide levels in surface waters for Scourge, Anvil and Altosid application.Jurnal of Shelfish Research 24.3 (October 2005).
[3] Laufer, Hans, et al. "Hormonal responses of lobsters to stress, an Interim Report" Third Long Island Sound Lobster Heath Symposium Program: 32-37.
[4] Walker, Anna, et al. "Metabolic effects of acute exposure to methoprene in the American lobster, Homarus americanus." Journal of Shellfish Research 24.3 (October 2005).
[5] Fraser, Doug. Cape lobster industry faces crisis (June 13, 2010) Cape Cod Times. http://www.capecodonline.com/apps/pbcs.dll/article?AID=/20100613/NEWS/6130340. Retrieved 02.27.12.

CLAIM: Mosquitoes are just like lobsters and methoprene will cause mortality in the same way.
TRUTH: If mosquitoes and lobsters are alike, then blue crabs (another related species) would experience the same problems in the presence of methoprene. They do not.

- In "*Estuary News*" (Vol. 16, No. 3, Spring, 2006), a publication of the EPA's *Delaware Bay National Estuary Program,* there appeared an article titled "*Protecting Horseshoe Crab and Human Health*" written by David Bushek of Rutgers University's Shellfish Research Laboratory. In the subsection boldly titled "Impact of Mosquito Control," it states that horseshoe crabs could be susceptible to adverse impacts from contaminants, and that "one contaminant of concern is the mosquito larvicide known as methoprene." The supposition was that methoprene prevents mosquito larvae from molting, and since horseshoe crabs are arthropods too, then according to the researchers' wisdom it would seem that methoprene might possibly contribute to the Bay's declining horseshoe crab population. The researchers exposed developing and newly-hatched larvae of horseshoe crabs to "environmentally relevant levels of methoprene." They reported, "After following development and survival through the first larval molt, we were unable to detect any macroscopic effects. That is the eggs and larvae appeared to develop normally, even at levels well above those applied to control mosquitoes along Delaware Bay."

CLAIM: Methoprene flows out of catch basins and into lobster-bearing waters.
TRUTH: Studies have consistently shown that methoprene, applied at recommended rates, is not flushed into natural waters in detectable amounts.

- Butler and Gettman investigated methoprene concentrations from the application of Altosid pellets into catch basins. The methodology included placing a known amount of pellets (3.5gm) into catch basins and subsequently determining the level of methoprene by chemical analysis. Three sampling points were used; in catch basins prior to flooding, at the outfall of the catch basins into Point Judith Pond, and 10 feet away from the outfall following flooding. A mesh trap was placed over the outfall pipe to determine if any of the pellets had washed out. Multiple replicate water samples were sent to two different laboratories for independent analysis.

  "Our results suggest that methoprene applied to catch basins in 30-day slow release pellets is not flushed into natural waters in detectable amounts ($\geq$ 0.2 ppb). Furthermore, our results suggest that the use of methoprene is not likely to affect non-target organisms in natural environments when used according to label instructions. Since methoprene concentrations were below detectable levels in Point Judith Pond, our study area, it is extremely unlikely that it would be found in larger bodies of water such as Long Island Sound or Narragansett Bay at levels anywhere near detection. Our data show that the only organisms likely to be exposed to methoprene at detectable levels ($\geq$ 0.2 ppb) are those that share catch basin environments with mosquito larvae."[6]

- In October 2009 and June 2010, the Rhode Island Department of Environmental Management performed additional studies to monitor the waters downstream from catch basins for the presence of methoprene. The 2009 study addressed Altosid levels (in ppb) from samples taken at an outfall following treatment and flushing of a series of 13 connected catch basins. None of

[6] Butler, Ginsberg, LeBrun, Gettman. Evaluation of nontarget effects of methoprene applied to catch basins for mosquito control. (August 2010) Journal of Vector Ecology.

the 11 samples yielded detectable levels of Altosid. These results are, again, indicative of the dilution factor, even though the experiment was designed to maximize the potential for acquiring detectable levels. The following year, in June 2010, an experiment addressed Altosid levels (in ppb) from samples taken downstream from an application in a tidal creek. 35 grams of pellets (representing the amount in 10 catch basins) were placed in a mesh pouch and secured at the bottom of a creek. One week later, during a gradual tidal flow, samples were taken adjacent to the pouch and at distances of 10, 20, and 30 feet downstream. None of the 12 samples yielded detectable levels of Altosid, again, indicative of the dilution factor.

- In a study conducted by Environment Canada in the Hamilton Ontario area, waters were sampled in areas where methoprene was actively used. Data shows that a low level of methoprene was seen in some samples closest to the treatment areas, but quickly disappears as it gets into streams and other bodies of water. Water samples were taken from inside the catch basins, and in only 50 percent of the basins were detections confirmed. The highest detection level was only sample at 4.35 ppb in a catch basin and the remaining detections were 0.65 ppb and lower. Receiving water samples from the catch basins were also collected and, generally, were determined to be below the concentrations of ecological concern as set by the interim Ontario provincial water quality objective (0.2 ppb). No methoprene was detected in Hamilton Bay.[7] Similarly, Fletcher, et al., did not detect methoprene in the Ontario receiving waters they tested.[8]

- Des Lauriers and colleagues concluded that rainfall flushed methoprene from the studied Toronto catch basins (the methoprene application sites) to the storm sewer outfall and into the receiving waters at concentrations lower than the level that may cause ecosystem damage.[9]

- The U.S. Geological Survey and U.S. Department of the Interior monitored surface waters in Suffolk County NY over a three year period for methoprene. Methoprene in excess of 1 ppb was found in a single sample at 9 ppb measured within an hour of application. No methoprene was found in four of the seven sites treated with methoprene.

- In a Washington State Department of Ecology study, methoprene was only detected in 6 percent (4 of 68 total) of the Grant County, Washington surface water samples Johnson and Kinney collected directly after methoprene treatment. Concentrations of methoprene ranged from 0.1 ppb to 0.6 ppb.[10]

- In response to discovery of West Nile Virus Beaufort County, SC, a costal county wit more water acreage than land, initiated a treatment regime. In response to environmental concerns about the harm to shrimp, crabs and oysters, the Beaufort County Mosquito Control initiated a study to determine the potential for catch basin treatment to result in methoprene concentrations

[7] Struger, John, et al. "Occurrence and Fate of Methoprene Compounds in Urban Areas of Southern Ontario, Canada." *Bulletin of Environmental Contamination and Toxicology* 79.2 (2007): 168-171.
[8] Fletcher, R., et al. "The Widespread use of Methoprene for the Prevention and Control of West Nile Virus in Ontario, Canada: Is it Impairing our Streams?" American Geophysical Union 86.18 (2005).
[9] Des Lauriers, Angelune, et al. "A field study of the use of methoprene for West Nile Virus mosquito control." Journal of Environmental Engineering and Science 5.6 (2006): 517-527.
[10] Johnson and Kinney. "Methoprene Concentrations in Surface Water Samples from Grant County Mosquito Control District No. 1." Washington State Department of Ecology Publication 06-03-001: (2006).

sufficient to disrupt the development of non-target species. Methoprene was found in 4 of 65 post-treatment water samples. The maximum concentration found was 0.285 ppb. The researchers concluded "Even with conditions believed to represent a worse-case scenario, catch basin treatment … did not result in methoprene concentrations sufficient to adversely impact non-target species. Further as methoprene degrades rapidly in the environment, accumulation over time to toxic concentrations is unlikely."[11]

CLAIM: Methoprene causes deformities in frogs.
TRUTH: Most of the concern adding fuel to this claim came from studies conducted with extremely high doses of methoprene (in some cases, 15,000 times the maximum recommended application rate).

- Methoprene was investigated as a possible cause for the reported increase in amphibian deformities. Most of the concern was generated by a study (La Clair et al. 1998) that reported that methoprene and its degradation products cause deformities in Xenopus laevis (frog embryo . However, not only were the studies conducted with extremely high doses of methoprene (ca. 15,000 times the maximum recommended application rate of Altosid) that had little, if any, relevance to methoprene in the environment, but the findings could not be replicated by other researchers. [12, 13]

- The extensive environmental studies that had already been carried out with methoprene, including specific studies on anurans (frogs)14 indicated that it was unlikely that methoprene or any of its degradation products were involved in the high rates of deformities observed.

- A study conducted by the EPA, (Degitz et al. 2003) evaluated the aqueous stability and developmental toxicity of methoprene and its degradation products in X. laevis embryos (FETAX). They found that methoprene exposure did not result in developmental toxicity at concentrations up to 2 ppb. They concluded that methoprene and its degradation products are not potent developmental toxicants in X. laevis and are not a factor in the occurrence of deformities in the environment.

- Ankley et al (1998), working for the EPA, conducted a study to evaluate the effects of both methoprene and ultraviolet light (UV) on the northern leopard frog (Rana pipiens) embryos. They found that UV light did cause limb abnormalities, whereas in the groups treated with methoprene only (up to 125 ppb), no increased mortality or developmental abnormalities were detected.

- In another study, Henrick et al. (2002) carried out a careful evaluation to address these concerns. Chemical analyses of water samples were carried out which demonstrated that methoprene and its degradation products could not be detected in the Minnesota water bodies where frog deformities were observed. A comparison of the use of Altosid in different counties

[11] Warren, D.A., et al. Assessment of Methoprene in Marine Waters after Catch Basin Treatment with Altosid XR Briquets.
[12] LaClair JJ, Bantle JA, Dumont J. Photoproducts and metabolites of a common insect growth regulator product developmental deformities in Xenopus Environ Sci Technol 32, 1453-1461 (1998)
[13] Degitz, S.J. et al. Devemopmental toxicity of methorene and several degradation products in Xenopus laevis. Aquatic Toxicology 64. 2003
[14] Miura and Takahashi 1973, Simonin et al. 1992, U.S. Environmental Protection Agency 2001.

in Minnesota with reported frog deformities showed no correlation between frog deformities and use of methoprene. This study also concluded that s-methoprene has negligible or no effect on anurans even at 100 times the maximum recommended field rate.

CLAIM: Methoprene was involved in the initial lobster die-off in Long Island Sound in 1999.
TRUTH: Methoprene was not used in the New York City mosquito program in 1999 since the effort that year focused on use of adulticidal controls. Altosid was not introduced in New York City until 2000.

- In 1999, West Nile virus was affecting the New York City area and as a result, the City of New York organized a concentrated mosquito control effort. This initial mosquito control effort was centered on the use of insecticides for adult mosquitoes. It was not until the following year, 2000, that Altosid was used in the New York City mosquito control program.

- A 2005 study in the *Journal of Shellfish Research* estimated the concentration of methoprene in Long Island Sound in 1999 based on the actual amount of methoprene used in the area. The model predicted extremely low levels, 0.0005 ppb methoprene, in the Sound. In this model, *all* of the methoprene applied in the catch basins of the watershed was predicted to reached the waters of Long Island Sound with no attenuation or decay — a highly unlikely scenario in practice. They concluded "the Phase I run for methoprene showed that even if all of the pesticide applied reached the water and never decayed, the concentration would not have been lethal to either larval or adult lobsters". [15]

- The surface waters of Long Island Sound were actually monitored in 2003 when methoprene was actively used in storm drains surrounding the Western Sound. No methoprene was detected. [16]

CLAIM: In 1999, lobsters were killed by mosquito pesticides; other factors had no effect.
TRUTH: A 2005 study published in the *Journal of Shellfish Research* points to a combination of factors that upset the balance of the lobster population in the Long Island Sound, including water temperature, disease, and increasingly hostile environmental conditions.

- "There is overwhelming evidence that in the absence of any pesticide application, a confluence and succession of factors pushed the balance of the WLIS lobster population far out of equilibrium with its environment in 1999. Faced with disease (paramoebae), and subjected to sustained and increasingly hostile environmental conditions, the immune systems of the lobsters were unable to compensate, and many lobsters succumbed. While the main mortality event occurred in 1999, the problem may have been building for several prior years.

- Sustained, above average water temperature was a driving force behind the snowball effect of environmental and oceanographic factors that stressed lobsters to the point at which their physiology and immune systems could not cope with these sustained and increasingly lethal environmental conditions in conjunction with, or in addition to, mounting an immune defense

[15] Landeck Miller, Robin, et al. "Application of water quality modeling technology to investigate the mortality of lobsters (Homarus americanus) in western Long Island Sound during the summer of 1999." *Journal of Shellfish Research* 24.3 (October 2005).
[16] Abbene, I.J. et al. Concentrations of Insecticides in Selected Surfac Water Bodies in Suffolk County, New Your, Before and After Mosquito Spraying, 2002-04.US Departoment of Interior US Geological Survey Open-File Report 2005-1384.

against the parasitic amoebae. Based on the body of evidence, the scenario leading to the 1999 mortality event is proposed as follows:

- Scattered reports of atypical lobster mortality began as early as 1997, however it is unknown when paramoebiasis began to affect the lobster population.

- In 1999, water temperature remained several degrees warmer than average for a duration of many months. For much of the summer and continuing into early fall, bottom water temperature remained at or above the temperature threshold that, in lobsters, induces a significantly enhanced respiration rate. The warm waters also likely accelerated the growth of natural populations of Neoparamoeba pemaquidensis in LIS waters.

- Lobsters in western LIS concentrated in numbers as they moved to deeper waters to escape warm shallower waters and moved away from hypoxia-affected areas towards those with slightly higher DO concentrations. This displacement, exacerbated by the abundant lobster population, caused crowding that, in the presence of less-than-optimal conditions, increased susceptibility to disease.

- In late August, winds from a front moving through the region completely mixed the water column in matter of hours, brought warm surface waters to the bottom, and raised warm temperatures several more degrees. While lobsters typically can endure abrupt changes in temperature, these lobsters were already in a weakened physiological state.

- Pesticides were being applied to combat the spread of WNV by mosquitos. Exposure of some lobsters in near-coastal waters to these pesticides could have resulted in sublethal effects, which may have further weakened those lobsters.

- As summer hypoxic conditions dissipated, low dissolved oxygen at the water sediment interface coupled with high water temperatures facilitated the release of sulfides and ammonium from the sediments, conditions shown to lead to a significant increase in the rate of mortality.

- Affliction with paramoebiasis sealed the fate of these lobsters. Either the energy used in mounting a sustained immune response to the disease took its toll on the lobsters, or the lobsters were unable to mount an adequate response as their immune systems were potentially compromised by the stressful conditions presented by this "perfect storm" of lethal synergistic effects. Mortality became rampant and the commercial lobster fishery in western LIS largely collapsed."[17]

CLAIM: Methoprene hasn't been studied in the Long Island Sound environment.
TRUTH: Multiple studies were conducted in the Long Island Sound in 2005 to examine the effects of methoprene on juvenile lobsters.

[17] Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (October 1, 2005). *Journal of Shellfish Research.*

- Studies by Zulkosky et al (2005) [18] and Miller et al (2005) [19] evaluated pesticide levels in the Long Island Sound following mosquito applications. Zulkosky evaluated effects of methoprene and adulticides on stage one and two larvae and juvenile lobsters. They also evaluated surface water concentrations following aerial applications of mosquito control products. Methoprene was found to have no effect on lobster larvae and juveniles at the highest dose tested of 10 ppb.

CLAIM: The problems with lobster populations in Rhode Island were related to methoprene.
TRUTH: Detailed analysis of abundance and mortality rate for several life stages of lobster in the Rhode Island area failed to reveal any evidence that the methoprene used in mosquito control programs has had any impact on the local lobster population.

- Methoprene is a growth disruptor for invertebrates that grow through molting and metamorphosis. It has the potential to kill and alter molting of larval lobster and may interfere with chitin deposition in shells of juveniles and adults (Walker 2005). The capable concentrations however are far above that likely to occur near the outfall of treated storm drains (Butler 2005). Moreover, the relatively short half-life of methoprene in water (Schaefer and Dupras 1973) and the enormous dilution factors operating on spatial scales of the fishery make it extremely unlikely that adverse impacts have occurred. In Black Point Narragansett, there was no evidence that abundance of settler, juvenile, or adult lobster was lower during years of methoprene usage (2000-2007) than years prior to usage (1990-1999).

- Lobsters in southern New England have experienced an epizootic outbreak of bacterial shell disease. It is likely that mortality rate in lobster has been increased by the disease (Gibson and Wahle 2005, Dominion 2008, ASMFC 2006). The outbreak however began in 1996, accelerated in 1997, and was well underway by 1999, before any methoprene treatments began in 2000. It is highly unlikely that methoprene had anything to do with a disease that still affects over 20 percent of the animals in the area. That is not to say that shell disease has not impacted the lobster population. It has likely killed some animals outright, induced abortive molts in some egg bearing females, and reduced growth rate in other animals. Still, with tens of thousands of animals examined the number dying remains relatively low at about 3 percent (Dominion 2008) and abortive molts are very rare and show no pattern with regard to methoprene usage (Figure 10). Continued study of the disease is warranted but it does not appear that methoprene is a strong candidate for new research. Altogether, the results do not support a methoprene impact hypothesis. Methoprene was similarly indicted in Long Island Sound as cause for a lobster die off but ultimately no evidence was found and increasing water temperatures were deemed more likely responsible (Dove et al. 2005).

- It is clear that the collapse of the Rhode Island inshore lobster stock and fishery in 2002-2004 and continued low abundance is not due to methoprene application. It occurred as a result of a "perfect storm" of failed settlement by the 1995-1996 cohorts, an acute mortality event (1996

[18] Zulkosky, A. et al. "Acute toxicity of resmiethrin, malathion and methoprene to larval and juvenile American lobsters (Homarus americanus) and analysis of pesticide levels in surface waters for Scourge, Anvil and Altosid application.Jurnal of Shelfish Research 24.3 (October 2005).
[19] Miller, Robin Landeck, et al. "Application of water quality modeling technology to investigate the mortality of lobsters (Homarus americanus) in western Long Island Sound during the summer of 1999." *Journal of Shellfish Research* 24.3 (October 2005).

oil spill) that winnowed extant juvenile populations, chronic shell disease, and overfishing (truncated age structure). The first factor was unavoidable, fishery managers cannot control long-term forcing by climatic events. The second event, while technically avoidable, was a result of human error that happens from time to time. The magnitude of shell disease as a mortality agent has yet to be fully understood but is believed low. Overfishing in the lobster fishery is given short thrift among all the factors that can be blamed. Despite the warnings of three peer-reviewed stock assessments (ASMFC 1996, 2002, 2006) it is still not appreciated how vulnerable a recruitment based fishery is.[20]

- An astonishing amount of methoprene would have to be applied in Narragansett Bay in Rhode Island to cause an effect in the lobster population. Narragansett would take an extraordinary quantity of methoprene applied to a watershed's storm sewer catch basins to reach harmful levels of the biopesticide in receiving waters. For example, the lobster harvest area in Rhode Island is approximately 87 square miles and would require more than 4400 metric tons of methoprene to be applied (assuming a water depth of only three feet). However, in 2007 only approximately 1.2 metric tons were sold in Rhode Island.[21]

CLAIM: It would be better to abandon the use of methoprene and just kill adult mosquitoes.
TRUTH: The opinion of the Suffolk County, New York superintendent responsible for mosquito control states, "By controlling mosquitoes in the larval stage, the use of methoprene products in Suffolk County prevents infestations that could adversely impact people while not exposing residents to pesticides. Preventing these infestations means there is less need for the use of the more toxic materials, adulticides, used to control adult mosquitoes. Not only are adulticides far more toxic to lobsters than methoprene, but their use can result in the exposure of County residents to these pesticides. Based on historic data, adulticiding by the County could increase 10-fold to achieve the level of control now achieved through larviciding with methoprene. In addition, pesticide applications by homeowners and other private interests could be expected to increase and these applications would involve doses 64x those used by Vector Control. This bill would eliminate a negligible risk to lobsters at the cost of increasing mosquito infestations, increasing the use of demonstrably more toxic materials and increasing the exposure of human beings to pesticides."

CLAIM: It would be better to only use Bti and avoid methoprene altogether.
TRUTH: In the Suffolk County Environmental Impact Study it states, one reason for the County to use multiple larvicide products is to allow for resistance management. The County tends to alternate between Bti and methoprene in salt marshes. Bti is effective on the younger growth stages of mosquito larvae, it is an ingestion toxicant meaning that larvae must eat it to receive a toxic dose. It provides another tool for mosquito control professionals to use but it cannot be relied on as a total larvicidal control method, especially in permanent water sources. Bti is susceptible to UV light degradation and competing food sources. It is less effective in higher organic matter environments and will only be effective for 48-72 hours in the water. As the season progresses, higher organics will develop in mosquito habitats which will lessen the effectiveness of the product. In situations with synchronous

[20] Gibson, Mark. Lobster Settlement and Abundance in Rhode Island: An Evaluation of Methoprene Application and Other Factors Potentially Influencing Early Survival. (June 2008) RI Department of Environmental Management, Division of Fish and Wildlife
[21] Based on data from "Rhode Island Commercial Fishing Areas" map produced by the University of Rhode Island Environmental Data Center, <http://www.edc.uri.edu/fish/imagemaps.html> (accessed June 16, 2008).

broods of mosquitoes, applications of Bti will be ineffective if applied to later stage mosquito larvae since these larvae do not feed.

Methoprene prevents larvae from developing, and is a contact pesticide and is effective on late stages. Since it works on the late stages, the younger mosquito larvae are left in the water column as a food source for beneficial species. Suffolk County records indicate large improvements in larval control effectiveness when methoprene was introduced in 1995.

CLAIM: Methoprene use endangers other organisms.
TRUTH: "Methoprene degrades rapidly in water so the use of most formulations in estuaries is generally not of concern. However, concern has in fact been raised in recent years with respect to methoprene's potential impact on shrimp, crabs and lobsters. These concerns stem from the fact that a shared evolutionary past, as well as resultant similarities in biology, exist between crustaceans and dipteran species (including mosquitoes).

Studies have indicated environmental and not chemical causes. Most of the recent studies of estuarine invertebrates have used shrimp, Atlantic oysters, amphipods, copepods, and mud crab. In general, impacts to these species are not anticipated to occur at expected environmental concentrations. The Caged Fish experiment found no impact to exposed fish and shrimp, for example. The risk analysis found no risks for impact to ecosystems because exposures were much less than the levels required to cause impacts to organisms. The lowest concentrations found to cause impacts to lobsters, when the exposures were sustained for days, were only recorded immediately after applications in local sampling.

This reinforces the conceptual understanding that the effectiveness of methoprene on mosquito larvae occurs at concentrations below those necessary to cause significant non-target organisms effects. Methoprene has been used by Suffolk County Vector Control SCVC since 1995, and is particularly useful in the salt marsh, where Bti is not always effective."[22]

CLAIM: Some scientists' work shows that methoprene is harmful to lobsters.
TRUTH: The primary concern with the use of methoprene (in salt marshes) appears to revolve around the work of two researchers, Horst and Hershey.

- Michael Horst has published research regarding impacts of methoprene on various crustaceans since 1999. He has found impacts, especially to larval stages of crabs and lobsters. Following are points to consider when reviewing his work, however, the Suffolk County is committed to continually reviewing ongoing research on all vector control pesticides and therefore, we will continue to evaluate any new data that becomes available in the future on methoprene.

  Studies conducted by Anne McElroy, PhD, Associate Professor and Graduate Program Director at the Marine Sciences Research Center at Stony Brook University, did not find methoprene lethal to larval lobsters at concentrations up to 10 ppb. In addition, she collected water from surface water bodies following vector control spraying by Suffolk County, New York and did not find the concentrations of methoprene to be acutely lethal in controlled laboratory setting. These studies by McElroy's laboratory are consistent with other published studies, which contradict the results of Horst's work.

Methoprene is applied in wetland areas, not where larval crabs and lobsters are found. Blue claw crabs hatch offshore and only arrive in estuaries when they are close to being fully developed. Lobsters hatch offshore, develop offshore, and live offshore. A modeling exercise, made to estimate the maximum amount of pesticides that could have been in Long Island Sound when the 1999 lobster die-off occurred, found the maximum amount of methoprene that could be present in the near offshore waters of the sound was measured in the parts per quadrillion, and the lowest concentration linked to effects are in the parts per billion.

Dr. Horst's 1999 research with crab larvae used concentrations up to 500 times higher than those levels present in real-world vector control applications. Dr. Horst's more recent work in 2005 with lobster larvae suggested that there was increased mortality utilizing concentrations of 1 to 2 ppb continuously during a 72 hour exposure. (These results were not confirmed in concurrent Stony Brook University analyses). In any case, 1 ppb methoprene for 72 hours is an extremely unrealistic exposure. The Caged Fish Study, conducted as part of the Long-Term Plan, clearly demonstrated that concentrations of methoprene rapidly decrease to 0.005 ppb within two hours of application.

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SECTION III: OTHER SUGGESTIONS FOR LOBSTER DECLINE IN NORTHEAST STATES

HOSTILE ENVIRONMENTAL CONDITIONS

"A spokeswoman for the New York Department of Environmental Conservation, Aphrodite Montalvo, said research shows the lobsters had been stressed by hostile environmental conditions and that individual pesticides were not enough to cause the die-off. She said the agency has upgraded sewage treatment plants and taken other steps toward improving water quality."
Conn. lobstermen seek other work as stocks dwindle. (2011, October 29). *Associated Press.*

PLASTICS

Researchers at the University of Connecticut have discovered waterborne chemicals leached from plastics and detergents, may contribute to the decline in population and to shell disease in lobsters in the Sound. Chemicals such as bisphenol A can slow the lobster's molting pattern and interfere with regular development.
Buckley, Christine. Lobster Dieoffs Linked to Chemicals in Plastics. (2010, August 10). *UConn Today Blog.*

"Hans Laufer, a research professor in the Department of Molecular and Cellular Biology in the College of Liberal Arts and Sciences, has found that by interfering with hormones crucial to young lobster growth, chemicals such as bisphenol A can slow the lobsters' molting patterns and interfere with regular development, leading to body deformations, susceptibility to disease, and potential death."
Buckley, Christine. Lobster Dieoffs Linked to Chemicals in Plastics. (2010, August 10). *UConn Today Blog.*

"Hans Laufer, a professor at the University of Connecticut, has spent the last four years investigating the link between the plastic byproducts, called alkyphenols, and shell rot. In recent years, the disease has become an epidemic in Long Island Sound (located between Connecticut and Long Island, N.Y.) affecting up to 70 per cent of some lobster populations at its peak. 'There seems to be a direct relationship between plastic compound breakdown and shell disease,' Laufer said. 'The lobsters try to molt out of the old shell,' Laufer said. 'If it's just mild enough, they recover. If it's serious, of course, it kills them.'"
Manzocco, Natalia. Steps taken to protect lobster; Fishing Group starts education program to warn of dangers of plastic in Bay of Fundy (2008, June 3) *The Telegraph-Journal.*

Climate Change/Water Temperature/Calcinosis/Shell Disease

"If Long Island Sound is becoming inhospitable for lobsters or other animals because water temperature is too high, that means they're not going to stay there and there's nothing anybody is going to do about it," said Gordon Colvin, the director of marine resources for the New York State Department of Environmental Conservation."
Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

''The correlation is very strong,'' [Dr. Alistair D. M. Dove] said. ''Not proven, but strong. Climate is the killer here.''
Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

''There are researchers who are sure it was pesticide -- and the lobster fishermen are sure -- but personally I now think there's very little chance,'' said Jack Mattice, the director of the New York Sea Grant Institute, which is helping oversee the lobster research. ''I believe that it was primarily temperature, and I think most people would probably say that temperature was a direct or indirect cause.''
Johnson, Kirk. Warming Waters and Dying Lobsters; Scientist Links Climate to Population's Decline in the Sound. (2002, November 9) *The New York Times.*

“Two summers ago, yet another lobster disease turned up. At first, it seemed just to add to the confusion. Orange grit was clogging and sometimes petrifying the gills of lobsters around eastern Long Island Sound. Under study, it proved to be deposited calcium, not unlike kidney stones in humans. It was called calcinosis. Then, its discoverer had a eureka moment. Alistair Dove, at the State University of New York-Stony Brook, got to thinking about what could drive a lobster’s metabolism and, by extension, cause such a metabolic disease. ‘That was the first time we thought of temperature,’ he says.”
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*

“Other scientists showed higher temperatures strain lobsters and may make them more susceptible to infection and pollutants. Maybe too much warmth is weakening their immunity to disease while making harmful microbes flourish, they reasoned. It could be luring warm-water predators into the Northeast and chasing cold-water ones from Maine. It could explain much of what lobstermen have encountered.”
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*

“Two scientific reports have shown that warming waters in the western Sound may have seriously contributed to the die-off. One report released this summer and associated with the Washington-based Union of Concerned Scientists (UCS) stated that "although a number of factors played a role in this die-off, warmer water temperatures seem to have set the stage."
Failoa, Anthony. What's Killing the Lobsters Of Long Island Sound? (2007, October 7) *The Washington Post*.

"What we found was that the concentrations of pesticides in the water could not have been high enough to be lethal to lobsters," said Sylvain De Guise, director of Connecticut's Sea Grant program and a lead researcher on a major 2005 study of the die-off published in the Journal of Shellfish Research. "Instead, we're probably looking at a combination of factors." He continued: "What you can say is that the western Sound is at the southernmost range for [coastal] lobsters, and it's very likely that the impact of warming waters would be seen here first. I'd have to say that global warming, based on common sense, is the strongest argument.""
Failoa, Anthony. What's Killing the Lobsters Of Long Island Sound? (2007, October 7) *The Washington Post*.

"' (The lobster decline) is a combination of factors that are all related back to changes in water temperature,' said Robert Glenn, a senior marine fisheries biologist with the state Division of Marine Fisheries.

Glenn said the expansion in the number of days with high water temperatures is probably a bigger factor than the temperature rise itself. While lobsters can tolerate the occasional warm day, prolonged exposure to water over 68 degrees wreaks havoc on their respiratory and immune systems and leads to outbreaks of shell disease and other lobster diseases."
Fraser, Doug. Cape lobster industry faces crisis (2010, June 13) ***Cape Cod Times.***

"Last year, a federal effort to coordinate research, the U.S. Global Change Research Program, found ocean warming already was forcing a migration of some species.

"The northward shifts we have seen in the area are due in part to climate change. We are starting to see some of the effects of global climate change in our area," said Janet Nye, a NOAA researcher working out of Woods Hole, Mass. She studied historical fish records and found that of 36 northwest Atlantic species, almost half had moved northward in 40 years as water temperatures warmed."
Struck, Doug. "Warming Waters Exacerbate Dwindling New England Fisheries." (2010, July 13). ***Scientific American.***

"It is not possible to draw a direct relationship between the decline of the Southern New England lobster stock and increased water temperatures. However, the strong coincidence in the timing of the increase in water temperature with the timing of the decline in landings, spawning stock biomass, and recruitment, coupled with overwhelming experimental evidence of increased physiological stress, immunosupression, and increased rates of disease in lobster exposed to prolonged periods of temperatures ≥ 20 °C, strongly suggest that increasing water temperatures have played a primary role."
American Lobster Technical Committee Atlantic States Marine Fisheries Commission. "Recruitment Failure in The Southern New England Lobster Stock." (2010, April 17) ***Accessed via Connecticut Department of Energy Environmental Protection web site.***

"In August 1999, the temperature differential between surface and bottom water temperatures in the Long Island Sound was as much as 5 [degrees]C and both nearshore waters and deep bottom waters continued to warm (CTDEP 1999, Wilson & Swanson 2005). On August 29th, the effects of Hurricane Dennis to the south of Long Island Sound, coupled with strong winds associated with a fast moving cold front from the north that passed through the LIS region, caused a complete vertical mixing of the water column, raising bottom water temperature several degrees to >22[degrees]C (Wilson et al. 2004, Wilson & Swanson 2005).

Following the passage of this front, there was, according to temperature and salinity data, limited restratification until September 16th, when Tropical Storm Floyd passed through the region, resulting in a substantial rainfall event and causing additional mixing of the water column. Water monitoring data for August through October indicate that bottom water temperatures remained elevated above 20[degrees]C (CTDEP 1999), a condition which Powers et al. (2004), Chang (2004), and Draxler et al. (2005) report induces respiratory stress in lobsters."
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (October 1, 2005) ***Journal of Shellfish Research.***

"Dr. [Carmela]Cuomo, [a marine ecologist and geologist at the University of New Haven] contended that even without the mixing event, the lobsters' long exposure to high water temperatures, low dissolved oxygen, ammonia and sulfides were enough to account for the 1999 die-off. She also warned that those conditions in Long Island Sound were far from rare. 'The probability of it happening again is very high,' she said."
Rather, John. Warm Water Killed the Lobsters. (2004, October 10). The New York Times.

"These warm water conditions may also have accelerated the growth of natural populations of the parasitic paramoeba, identified as Neoparamoeba pemaquidensis, found infecting the lobsters in 1999 (Mullen et al. 2005). In late October, more than 90% of lobsters examined were infected with paramoebae; those collected later in the fall had infection rates of about 29% (P. Howell, CTDEP, pers. comm.). This paramoeba inhabited LIS prior to 1999 (Mullen et al. 2005), therefore, it is possible that paramoebiasis was beginning to infect the lobster population prior to 1999.

Unfortunately none of the lobsters that died in 1997 or 1998 was set aside for a pathologic work-up. Strains of N. pemaquidensis are facultative pathogens of sea urchins (affecting the nerve ring), salmon and, now, American lobsters (Mullen et al. 2005). The presence of paramoebae in lobsters was monitored for 3 years following the 1999 die-off; more than 800 lobsters collected systematically throughout the Sound by CTDEP and examined by pathologists at the University of Connecticut exhibited infection rates of 0% to 14% (P. Howell, CTDEP, pers. comm.)"
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (October 1, 2005) ***Journal of Shellfish Research.***

HYPOXIA (i.e., LACK OF OXYGEN)

"Since the late 1980s, the CTDEP Water Management Bureau has monitored water quality throughout the Sound. Dissolved oxygen data are used to assess the severity and extent of hypoxic conditions that develop in late summer in western LIS, which in 1999 were not especially severe (Gates 2000). Hypoxic conditions were present from July 2nd through August 21st, covering about 120 sq. miles (http://dep.state.ct.us/wtr/lis/monitoring/ summer99.htm). Hypoxic conditions were most severe during the first week of August. Data also showed a strong correlation between elevated bottom water temperatures and low bottom water dissolved oxygen levels in a west-to-east gradient across the Sound.

Three years of survey work by CTDEP in the early 1990s produced a response curve for lobsters with respect to hypoxic areas. Lobsters were found to "herd" or crowd in greater numbers near margins of hypoxic zones where dissolved oxygen (DO) concentrations were >2 mg/L, and leave areas where DO was [less than or equal to]2 mg/L (Simpson 2000). This suggests that as the lobsters moved from shallow to deeper waters to find cooler water in summer 1999, they were at the same time avoiding severely hypoxic areas, with the result that the lobster population was further concentrated in certain regions of western LIS."
Pearce, Jack; Balcom, Nancy. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. (October 1, 2005) ***Journal of Shellfish Research.***

OVERFISHING

"By official standards, lobstermen are overfishing Northeastern waters. They are leaving too few lobsters to breed later generations, even in Maine, according to calculations of government biologists. Some predict drastic decline in Maine's future."
Donn, Jeff. Northeast lobster decline tied to warming. (2004, August 24). *Associated Press.*

"Biologists on a multi-state Fisheries Commission committee have found that warmer waters, disease and fishing have depleted lobster stocks, and they recently recommended a five-year ban on lobstering from Cape Cod to Virginia."
Struck, Doug. "Warming Waters Exacerbate Dwindling New England Fisheries." (2010, July 13). *Scientific American.*

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